

AD 680495

TRANSLATION NO. 30 d

DATE: July 1968

DDC AVAILABILITY NOTICE

This document is subject to special export controls and each transmittal to foreign governments or foreign nationals may be made only with prior approval of commanding Officer Fort Detrick, ATTN: SMOF/AE-T. Frederick, Md. 21701.

JAN 17 1969

DEPARTMENT OF THE ARMY  
Fort Detrick  
Frederick, Maryland

Approved by the  
CLEARINGHOUSE  
for Federal Scientific & Technical  
Information Springfield Va. 22151

On the pathological arterial changes in the spleen.

by A. Nakonetschny.

---

Virchow's Archives, 245: 564-577 (1923).

---

In view of the latest concepts relative to the nature and genesis of atherosclerosis, it becomes necessary to examine those histological changes of the various arteries which even today are still grouped under the collective name of arteriosclerosis by many authors. The frequent changes of the splenic arteries are of particular interest at this time and it may be asked which peculiarities they reveal in comparison with other atherosclerotic arterial changes.

It will be my task in the present paper to describe the pathological alterations characteristic for the splenic arteries, and to give a picture of their gradual inception.

The special literature on this problem is sparse. Particular attention is directed to Herxheimer's work, who stresses the frequent hyaline degeneration of the splenic arteries. According to his description, the walls of the small splenic arteries are strongly thickened and swollen, the hyaline masses now encompass the entire diameter of the vessel, now only a portion. No cell nuclei are demonstrable in the hyaline masses. The endothelium frequently is separated. The vascular lumen is considerably narrowed, even up to complete closure.

The author does not treat the question of fatty deposits and hyperplastic changes of the splenic arteries in detail. He does not want to include the vascular changes described by him among atheroscleroses and explains them as "a functional manifestation of detrition or adjustment." Eppinger also found hyaline deposits in the intima of small splenic arteries in 3/4 of all cases examined by him, and considers them typical changes in pernicious anemia and hemolytic icterus.

In 1922 Matsuno described the process of the fibrous intimal thickening of small splenic arteries, commencing in youth and complicated in later years by secondary, regressive manifestations. This leads to "atherosclerosis," developing in the wake of functional attrition.

Of the earlier authors, Stilling gives us more precise indications of the hyaline degeneration of splenic arteries. This author thinks that hyaline is deposited primarily in the media, but often also in the adventitia. The author says: "I invariably found the epithelium and intima to be unchanged."

In contrast, Mallat points out that hyaline deposits occur in the intima as well as the media. Klein also observed a "hyaline metamorphosis of the arterial vessels" up to complete closure of the latter's lumen.

Other authors (Ssoznitschewski, Leven, Prym, Jores, Fahr) have made only incidental references to the changes of splenic arteries, particularly in connection with the changes of renal arteries in contracted kidney. In this connection most of these authors tend to identify the changes of the smaller arteries of the internal organs, also of the spleen, with a manifestation of the general picture of pathological alterations in the vascular system. As early as 1880 and 1892 Ssoznitschewski and Leven pointed to a connection between the changes of the splenic arteries and the changes of the entire vascular system. The latter describes a complete fibrous induration of the spleen and considers it a result of vascular disease, the vessels being, in his words, "highly arteriosclerotic." The author did not describe the nature of this arteriosclerosis, however. It is only since the publication of Jores' papers that added attention is devoted to the finer structure of the affected arterial wall, also of the splenic arteries, and that the importance of elastic-hyperplastic intimal thickening is being stressed (Jores, Fahr).

In 1920 Hueck has drawn a sharp line between hyaline degeneration and other pathological processes observed in arteriosclerosis. He ascribes the cause of hyaline degeneration to a loosening of the intima and to certain chemical, perhaps also fermentative factors which promote the deposit of hyaline, the chemical structure of which is still unknown. (\*)

Prym treats the phenomenon of fatty deposits in the arterial walls in more detail, particularly those of the spleen. His description indicates that the intima becomes fatty especially in the small splenic arteries, and this not uniformly, but in the form of button-shaped thickenings (on cross sections). On longitudinal sections these thickenings occur as fatty aggregates in the arterial wall, where they protrude toward the media and compress the latter. Owing to this circumstance the author was unable to establish with certainty whether fat was deposited only in the intima or also in the media. He received the impression that the media as well as the adventitia were included in this fatty process. According to his description, severe fatty degeneration leads to a complete obliteration of the small arteries. "Fatty thrombi" are frequently found in the latter's lumen. The author concludes generally that fatty deposits occur along the elastic fibers; he observed in the initial stages that they start at the lum. elast. int.

Jores also points to the fatty degeneration of the small splenic arteries and lists them in the records of the material tested by him. The small splenic arteries were invariably involved, usually severely so; the follicular arteries were, as a rule, affected by severe degeneration, the larger arteries with or without commencing degeneration gave the appearance of severe elastic-hyperplastic intimal thickening.

\*\*\*\*\*

(\*) Herm. Fischer's "Contributions to the problem of hyaline degeneration of the small splenic vessels," Inaug. Diss. Munich 1919, unfortunately was not accessible to me.

Kusunoki also treats the problem of fatty degeneration of splenic arteries; he also observed fatty deposits in the trabeculae and the capsule. In some cases he found lipoid grains in the vascular lumen and in all wall layers of the small vessels. Severe fatty degeneration was frequently noted in the intima and occasionally in the adventitia. In certain cases the focal fatty infiltrations were particularly pronounced in the connective tissue bundles and the elastic fibers of the thickened adventitia. The lipoid deposit in the intima of the small splenic arteries is often so severe in arteriosclerosis that the lumen is obstructed.

During the general compilation of my material (see table) I noted that here, too, the three groups of splenic arterial changes described in the literature were prevalent: 1. hyperplastic processes, 2. hyaline deposits and 3. deposit of lipoid substances.

In the following, these 3 groups shall be discussed individually.

I have examined a total of 50 spleens. The material was obtained from the Peter-Paul Hospital at St. Petersburg and included corpses ranging in age from 5 months to 69 years. Those cases revealing a pronounced intimal thickening of the aorta of varying degree and of fatty or fibrous character, were preferred. My designation of splenic arteries is patterned on Meidenreich, who differentiates 1. trabecular arteries, 2. central arteries, 3. pulpar arteries and 4. branches of the penicillus. The latter 3 groups showed very similar changes in my material and therefore will be consolidated at the appropriate places under the designation "small splenic arteries." In some cases the Art. lienalis was also examined for reasons of comparison.

The post mortem material was fixed in Orth's mixture or in formalin. The sections were prepared with the freeze-microtome and the following stains: Van Gieson, Weigert (elastic tissue). Sudan III. Hematoxylin eosin, gentian violet (amyloid).

#### Hyperplastic changes of the splenic arteries.

At age 5-6 months (case 1,2) the elastic stain of cross sections of trabecular arteries shows the following picture: The endothelium is covered on the outside by a strongly serpentine Lam. elast. int. The latter is seen on cross sections as densely packed points, consisting therefore of a row of longitudinal elastic fibers. The media is composed of 3-4 rows of circularly oriented smooth muscle fibers. Between these there are very fine serpentine elastic fibers, also with circular courses. Outward from the media is the weakly pronounced Lam. elast. ext. The narrow, fibrous adventitia reveals only a few elastic fibers.

In the 2d year (case 3,4) the microscopic appearance of the trabecular arteries is changed but little: The Lam. elast. int. is more pronounced and its disrupted character is even more distinct on cross sections. The Lam. elast. ext., also consisting of a row of longitudinal elastic fibers, is also more evident. The elastic network of the adventitia is still

indistinct at this age, but may be recognized in places. Generally, the microscopic appearance of the trabecular arteries does not show noteworthy changes up to the 20th year. In later years the wall layers of these arteries become thicker; the Lam. elast. int. becomes wider, copious elastic fibers appear in the media, the elastic network of the adventitia becomes denser, the individual fibers thicker.

I was able to demonstrate a diffusion of the Lam. elast. int. in the trabecular arteries only in the 38th year (No. 14, 15). The longitudinal section of a trabecular artery of case 15 shows the following: There is a second, newly formed lamella located on the inside of the Lam. elast. int. The interval between these two lamellae is occupied by longitudinal nuclei of muscle fibers. The elastic fibers of the adventitia form a dense network.

I observed this intimal thickening almost without exception in older material (No. 16-50), and its intensity usually increases with advancing age. Thus the intima of a trabecular artery of a 56-year-old man (case 35) consists of 2, in places of 3 lamellae which apparently have developed by a split in the Lam. elast. int. There are longitudinal muscle fibers between these elastic lamellae. The media and adventitia show the picture described above.

The central and pulpar arteries show few differences and will therefore be discussed together.

In children aged 5-6 months (No. 1 and 2), longitudinal sections of the arteries show a thin but distinctly pronounced homogeneous Lam. elast. int. under the endothelium. On cross sections it reveals a serpentine course and consists of a row of densely packed longitudinal fibers. The adventitia has the same structure as in the trabecular arteries, the Lam. elast. int. is even less distinct.

In the second year (case 3,4) the structure of the central and pulpar arteries just described becomes more distinct. At this age some arteries of this type fail to show any Lam. elast. ext., and the muscle fibers of the media are surrounded only by a delicate network of elastic and collagenous fibers.

With advancing age the Lam. elast. int., as already described in connection with the trabecular arteries, becomes thicker and is expressed as a distinct interrupted line on cross sections. The elastic network and the collagenous fibers of the adventitia also become more pronounced. The media gives evidence of an increase in muscle fibers, now deposited in 2-3 rows. A cleavage or splitting of the Lam. elast. int. could not be demonstrated with certainty in this group of arteries.

Concerning the penicillus branches, they showed only an increase in elastic fibers of the adventitia with advancing age.

Art. lienalis. On cross sections of the Art. lienalis of children aged 5-6 months (case 1,2), the Lam. elast. int. is situated next to the endothelium in the form of a homogeneous, circular lamella. The media consists of 12-15 rows of circular muscle fibers with wavy, also circular elastic fibers between them.

The adventitia is broader than the media. In its outer layers the elastic fibers are looser and with circular courses on cross sections (case 2). In the inner part of the adventitia the elastic fibers of the same section are more densely packed, interrupted, and seem to be composed of individual points and rods; we are therefore dealing with a network of elastic fibers that are cut both crosswise and at an angle. Between these elastic fibers there are connective tissue cells and fibers running in every direction. A Lam. elast. ext. is not always evident on the border of the media.

In the second year (case 3,4) the cross section of the Art. lienalis shows the following picture: A short portion of the vascular diameter reveals a distinct cleavage of the Lam. elast. int. into 2 lamellae. There are longitudinal smooth muscle fibers between the latter. The media shows the same structure as in the previous case. The adventitia is almost as wide as the media. The elastic network is denser, more compact in the inner layers of the adventitia. The Lam. elast. ext. is not present throughout the entire vascular circumference.

At an advanced age a partial cleavage of the Lam. elast. int. is invariably present, now more distinct, now more weakly pronounced. In the 35th year (case 35) the Lam. elast. int. of the Art. lienalis is already split along the entire vascular circumference on cross sections. At some places of the vascular circumference, 2-3 rows of smooth, longitudinal muscle fibers are occupying the space between the two lamellae; i.e. a Joresian musculo-elastic layer. In one part of the vascular circumference there are also newly separated lamellae and fibers on the inside of the layer, resulting in occasionally quite considerable intimal thickening of an elastic-hyperplastic character. The media has more elastic fibers than heretofore. The elastic network of the adventitia is directly proximal to the media in form of a narrow wreath. A Lam. elast. ext. cannot be demonstrated.

With advancing age this appearance of a muscular-elastic and elastic-hyperplastic intimal thickening is regularly repeated in every case and distinctly gains in intensity. On the cross section of the Art. lienalis of a 38-year-old man (case 15) there is a thickening of the intima, irregular in width and located inside of the cleft L. elast. int., composed of a network of connective tissue fibers and thin, elastic fibers that anastomose with a predominantly circular orientation. The latter apparently have no connection with the Lam. elast. int. (the connective tissue layer of Jores).

After the 40th year the intimal thickening just described, the connective tissue layer, is seen frequently in the Art. lienalis, and sometimes

takes on a pronounced form (case 18,45,46). The connective tissue layer either borders directly on the Lam. elast. int. or on the elastic-hyperplastic or muscular-elastic layer.

Examination of the hyperplastic changes of the splenic arteries lead me to the following conclusions:

1. In the splenic arteries the hyperplastic changes increase constantly with advancing age, commencing in the first year of life.

2. The hyperplastic changes of the small arteries are expressed in the increasing enlargement of the Lam. elast. int. and the heightened development of the elastic and collagenous fibers of the adventitia.

3. The Lam. elast. int. of the trabecular arteries widens with increasing age. In middle age a cleavage of the Lam. elast. int. may occur, as well as a development of a muscular-elastic and elastic-hyperplastic layer, also increasing with advancing age. The elastic network of the adventitia becomes thicker with advancing age; the individual fibers move closer together. The quantity of circular elastic and muscular fibers in the media also increases.

4. The hyperplastic changes of the Art. lienalis start already in childhood (cleavage of the Lam. elast. int.) and may reveal the development of all 3 Joresian layers in the thickened intima in middle age.

## 2. Socalled "hyaline degeneration" of the splenic arteries.

In examining the changes of the splenic arteries, I was able to demonstrate homogeneous masses on the inside of the media in 35 out of 50 cases, giving a yellow reaction to van Gieson and a negative amyloid; they therefore consisted of deposits of socalled hyaline.

Depending on the intensity of the changes caused by these deposits, 3 degrees of hyalinosis may be differentiated microscopically in the splenic arteries:

The 1st degree: Inward from the media and Lam. elast. int. there are homogeneous masses stained yellow by van Gieson. These now envelope the entire vascular circumference and form a narrow circle, now lie in irregular masses in various parts of the vascular circumference. The media is pushed slightly outward, but retains the regular arrangement of muscle fibers. Toward the lumen the hyaline masses are directly covered by the endothelium. The Lam. elast. int. is expanded and has lost its wavy form. The adventitia is unchanged in comparison to cases without hyaline.

The 2d degree: The vascular lumen is distinctly narrowed and surrounded by a wide ring of compact, homogeneous masses. The media is considerably narrowed and displaced outward. The muscle fibers lose the

regular character of their circular course. The Lam. elast. int. loses its wavy form, is expanded, displaced outward. In places it seems fragmented and occasionally cannot be seen at all in its entirety; only isolated fragments are recognized.

The 3rd degree: The vascular lumen is considerably narrowed. The individual wall layers of the arteries can no longer be differentiated. They appear in the form of a wide ring of homogeneous masses with a barely visible lumen in the center. Occasionally, isolated cell nuclei may be found in these homogeneous masses, evidently of muscle cells. The Lam. elast. int. cannot be seen at all, nor can the elastic network of the adventitia be demonstrated in many cases.

All 3 degrees of change in the splenic arteries caused by hyaline deposits may be observed in the same case. Frequently a severely changed vessel is seen next to one with an unimpaired structure.

Hyaline degeneration afflicts primarily the branches of the penicillus, followed by the pulpar arteries and central arteries. Hyaline deposits in the trabecular arteries are considerably rarer and less extensive (case 37, 18 and others).

My material gave evidence of hyaline deposits in the splenic arteries in early childhood -- in children 5, 8 and 12 years old. It must be noted that up to about the 20th year hyaline is found usually in isolated arteries of the spleen (case 5-9); after the 20th year we usually see extensive hyaline degeneration of many arteries in the majority of cases. The degree of degeneration does not always correspond to the age, however.

I have been unable to discover a relationship between the symptom of hyaline deposits and the type of disease (cf. also the papers of Herxheimer and Matsuno).

The results obtained relative to the hyalinosis of the splenic arteries may be summarized as follows:

1. The "hyaline degeneration" of splenic arteries is a very frequent occurrence and begins in early childhood.
2. The deposit of hyaline commences in the intima of the small splenic arteries, inward from the Lam. elast. int. With increasing hyaline deposits the Lam. elast. int. is displaced outward, becomes fragmented and finally disappears altogether. The media is also displaced outward and atrophies.
3. Fatty deposits in the splenic arteries.

The material tested by me frequently revealed fatty deposits in the splenic arteries, occurring in 3 different types.

Type 1: The fatty deposits are found preferably in the homogeneous hyaline masses of the arterial wall. Usually fat may already be found in the walls at a youthful age (case 5-10), at which time they contain only scanty hyaline (Fig. 1). No fat is found in the media and adventitia.

We see the most diverse pictures in the arteries with more strongly pronounced hyaline degeneration (case 17, 23, 29, 42, 47 and others), depending on the degree of fatty deposit. Thus arteries are seen with large masses of hyaline and only scant amounts of fat in the form of isolated drops, small diffusely distributed masses or finely grained deposits. At times the fat is distributed in foci, sometimes it envelopes the entire vascular circumference. Frequently the fat is deposited only in the outer layers of the hyaline masses proximal to the media (Fig. 2). The part of these masses turned toward the lumen contains no fat (case 35, 37). Finally, arteries are found in which the entire hyaline mass is completely permeated with fat. Cross sections of such arteries show a wide wreath of fatty masses around the lumen; the latter may even be closed completely thereby (case 39, 49 and others).

In all variations of fatty deposits in arteries with distinctly pronounced hyaline degeneration discussed so far, the media is more or less distinctly visible and fatty aggregates are seen at the nuclear poles of their muscle cells, or the media has become completely unrecognizable in its structure owing to the hyaline masses deposited in the vascular wall (see above). Less frequently such cases also reveal fatty deposits in the adventitia, in the form of fatty aggregates along the elastic fibers.

This type of fatty deposit was seen in my material starting with the 5th year, i.e. from a point when hyaline degeneration was already present. The fat is deposited secondarily, and this only in the arteries where hyaline is already present, i.e. preferably in the small arteries, in the branches of the penicillus, the pulpar arteries and the central arteries.

Type 2: Here the fat is deposited primarily along the elastic membranes and fibers of the intima, especially in the trabecular arteries. Microscopic examination reveals a serpentine fat line inward from the media, corresponding to the progression of the lam. elast. int., now surrounding the lumen in the shape of a ring, now occupying only a part of the vascular circumference (Fig. 3, case 38, 40 and others). In the media, fat drops are usually demonstrable at the nuclear poles of the muscle cells. The adventitia reveals only small amounts of fat along the elastic fibers.

This type of fatty deposit is found also, though not exactly with frequency, in the central arteries and the pulpar arteries, in which no hyaline is demonstrable (case 38, 47). If hyperplastic intimal thickening of the trabecular arteries is present, the fat is deposited along all elastic fibers of the hyperplastic intimal section.

I observed this type of fatty degeneration of the splenic arteries mentioned in my material starting from the 40th year (case 14,18). It is a frequent occurrence in advanced age. This type may be considered to be characteristic for the Art. lienalis and it appears here simultaneously with the cleavage of the Lam. elast. int., i.e. already in the 2d year, judging from my material (cf. above -- hyperplastic changes of the splenic arteries).

Type 3 (Fig. 4 and 5). Here the fat is diffusely deposited in all layers of the vascular wall of the splenic arteries, although it prefers the adventitia. This type of fatty degeneration is seen most often in the trabecular arteries, but it occurs also in the smaller arteries.

It is worth mentioning that in these cases there is frequently a simultaneous pronounced fatty degeneration of the capsule and the trabeculae, following the elastic fibers of the deeper capsular layers and the trabeculae. In all such cases copious amounts of fat are found also in the protoplasm of the pulpar cells, apparently these are reticulo-endothelial cells. In the arterial wall it is primarily the adventitia that is affected by fatty deposits in these cases (case 14,30,42,50 and others). Here the fat is also concentrated along the elastic fibers. In the media the fat is usually seen as small drops at the nuclear poles of muscle cells. In the intima the fat deposit follows the elastic fibers (preferably in the trabecular arteries)(Fig. 4), or it takes place in the hyaline masses if such are present (case 42 and others; small splenic arteries).

The last type of fatty deposit was rarer among my material than the two types described previously, and this only after the 40th year. It was noted further in some of these cases that the blood plasma in the arteries gave an orange-colored hue after staining with Sudan III (case 29,32,39 and others).

The 3 types of fatty degeneration described by us do not represent isolated, strictly delineated forms, as they occur in all possible combinations. Thus, for instance, the same case will often reveal small arteries with fatty deposits in the hyaline masses, but intimal fatty degeneration in the trabecular arteries (type 1 and 2). The simultaneous fatty deposit in the hyaline masses and along the elastic fibers of the intima in diffuse splenic fatty degeneration (type 3) has already been mentioned above.

Generally, it must be said that fatty deposits in the splenic arteries occur quite frequently and were demonstrable in various degrees of severity and form at a more mature age in almost all of the spleens examined by me. Fat is present in the hyaline masses at an early age, even in children; the other types appear only at a more mature age, but then in almost all cases.

The changes in the splenic arteries observed by me may be classified in 2 groups. To group 1 belong all hyperplastic changes of the larger

splenic arteries (trabecular arteries, less frequently central arteries), which increase regularly with advancing age. These changes apparently are a common senile manifestation and depend on the increased demands on the functional performance of the vascular system (Merxheimer, Mitsuno), which increase with age; this being applicable also to the other arteries, e.g. the coronary vessels of the heart (Wolkoff). In later years these hyperplastic changes are connected also with the hyaline degeneration of the smaller splenic arteries, since a considerable impediment to the blood circulation apparently develops in this case. Similar conditions are found also in the kidneys (Fahr, Benda and others).

Thus the hyperplastic changes of the splenic arteries, which apparently represent a functional adaptation, must be strictly delineated from the other pathological processes observed here. Of the latter, we are dealing in the spleen primarily with hyaline and fatty deposits. These two processes are similar in that substances appear in the arterial wall in both cases which do not represent decomposition products of the elements contained in the arterial wall, but are primarily deposited between them. However, the localization and nature of these deposits are radically different.

The hyaline deposit represents an independent process, which customarily affects only the small arteries of the internal organs and which must be sharply separated from atherosclerosis as conceived by Marchand, Aschoff and others; this form of hyalinosis has no part in the atherosclerosis of the large arteries and does not evoke secondary hyperplastic processes in the intima.

Concerning fatty deposits in the splenic arteries, that form marked by isolated fatty aggregates along the elastic fibers of the intima (trabecular arteries) is completely analogous to the intimal fatty degeneration of the large vessels, i.e. to the form which is characteristic also for the A. lienalis (see above). This process cannot be completely identified with atherosclerosis, however, since the secondary hyperplastic changes and cell reactions are absent from the splenic arteries, and it seems more appropriate to speak of simple "fatty degeneration." The 3rd type is similarly marked by fatty deposits along the elastic fibers, but here this process is spread diffusely over the entire elastic fiber network of the stroma and the other splenic elements. The fact that some of the cases discussed above gave evidence of a microscopically distinct lipemia, offers additional interest. This circumstance may possibly indicate the etiology of this type of fatty degeneration, which then would be found in an increased lipid content of the blood.

With the 1st type of fatty deposit we are finally dealing with secondary fatty degeneration of the hyaline masses, as observed also in connection with amyloid; I was able to confirm this in one case of amyloid spleen (case 20). This type is interesting to the extent that here the significance of local conditions for the deposit of fat is clearly indicated. The physical-chemical conditions presumably present in the hyaline masses are very favorable to the settling of fatty masses supplied from the outside,

and this condition is probably to blame for the fatty degeneration of the hyaline masses occurring at such an early stage.

The deposit of fat in the hyaline masses and in the intermediary substance, along the elastic fibers so frequently observed, are processes closely related in their nature. Apparently a suitable substance is involved in which the fat precipitates easily, as is the case also in organized thrombi (Zinserling).

Finally, the difference between the alternative types of the trabecular arteries and those of the small splenic arteries deserves to be pointed out (see above). The trabecular arteries reveal processes that approximate those of the A. lienalis and that simulate the well-known appearance of atherosclerosis. However, all the changes characteristic of atherosclerosis, such as the aggregation of lipid-containing cells, fibrous intimal thickening, secondary calcium deposits, cannot be demonstrated here.

#### Illustrations

Fig. 1. Central artery in the spleen of a 6-year-old child (case 7). Hyaline deposit with fatty degeneration. (Fatty process type 1). Stain of all preparations with hematoxylin-Sudan III.

Fig. 2. Central artery in the spleen of an adult (case 19). High grade deposit of hyaline with scant fatty degeneration (fatty process type 1).

Fig. 3. Central artery (case 38). Fatty degeneration of the inner elastic lamella (type 2).

Fig. 4. Trabecular artery in longitudinal section (case 47). Fatty degeneration of the inner elastic lamella and the elastic fibers of the adventitia. Hyperplastic intimal thickening.

Fig. 5. (case 50). Diffuse type of fatty degeneration. High grade fatty degeneration of the inner elastic lamella in a trabecular artery and the walls of the central arteries and a few pulpar cells. Diffuse fatty degeneration of the fibrous tissue of the trabeculae.

Table 1.  
(the number of crosses represents the degree of alterative severity).

Nr.	Age & sex	Anatomical diagnosis	Hyperplastic changes	Fatty deposits		
				Hyaline deposits (small arteries)	in the hyaline masses (sm. arteries)	along the elast. intimal fibers
1	5 mo M	High grade inanition	-	-	-	-
2	6 mo F	Inanition	-	-	-	-
3	2 yr F	Dysentery	-	-	-	-
4	2 yr F	Inanition. Pulmonary phthisis	-	-	-	-
5	5 yr M	Inanition. Dysentery	-	+	+	-
6	5 yr F	Inanition. Purulent pneumonia	-	+	+	-
7	8 yr F	Inanition. Pulmonary phthisis	-	+	+	-
8	10 yr M	Miliary TB. Tuberc. meningitis	-	+	+	-
9	12 yr F	Pulm. phthisis. Intestinal TB	-	+	+	-
10	19 yr M	Inanition. Septicopyemia	-	++	++	-
11	21 yr F	Heart disease. Embolism of pulmonary artery	-	-	-	-
12	26 yr F	Dysentery. Perfor. peritonitis	-	++	+	-
13	35 yr F	Dysentery	-	-	-	-
14	38 yr F	Stomach cancer. Atherosclerosis	+	+	+	++
15	38 yr M	Ileus	+	++	+	+
16	40 yr F	Pleur. empyema. Dysentery	+	++	+	-
17	40 yr F	Stomach tumor. Suppurative peritonitis	-	+++	+++	+
18	40 yr F	Cardiac callosity. Atherosclerosis. Contr. kidney	+	+++	++	+
19	41 yr M	Pulmonary phthisis	+	+++	++	+
20	42 yr M	Pulmonary phthisis. Nephrosis	+	-	-	-
21	42 yr F	Lobar pneumonia	-	-	-	-
22	47 yr M	Purulent hepatic echinococcus. Septicopyemia	-	+	-	+
23	47 yr M	Inanition. Relapsing fever	-	++	++	++
24	49 yr F	Pulm. phthisis. Atherosclerosis	+	+++	++	+
25	50 yr M	High grade atherosclerosis. Pulmonary phthisis	++	++	+	-
26	50 yr F	Inanition. Pulm. phthisis. Bronchopneumonia	+	-	-	-
27	50 yr M	Stomach cancer	+	++	+	-
28	51 yr F	Atherosclerosis. Cerebral hemorrh. Contr. kidney	+	++	+++	+

No.	Age & sex	Anatomical diagnosis	Fatty deposits				
			Hyperplastic changes	Hyaline deposits (small arteries)	in the hyaline masses (sm. arteries)	along the elast. intimal fibers	diffuse
29	51 yr M	Atherosclerosis. Lobar pneumonia	++	+++	+++	+	-
30	52 yr F	Inanition. Dysentery. Atherosclerosis	-	++	+	+	+
31	52 yr F	Meningeal endothelioma. Atherosclerosis	++	+++	+	+	+
32	53 yr M	Relapsing fever	-	++	++	++	+
33	55 yr M	Atherosclerosis. Cardiac callosity	+	+++	+	+	-
34	56 yr F	Stomach cancer. Purulent pneumonia	+	++	+	-	-
35	56 yr M	Stomach cancer	+++	+++	++	+	+
36	56 yr M	Relapsing fever	-	-	-	-	-
37	57 yr F	Atherosclerosis. Contracted kidney. Bronchopneumonia	++	+++	+++	++	+
38	59 yr M	Coronary sclerosis. Cardiac insufficiency	++	++	+	+	+++
39	59 yr F	Atherosclerosis. Lobar pneumonia	+	+++	+++	+	++
40	60 yr F	Dysentery	++	+	+	++	++
41	62 yr F	Acute glomerulonephritis	-	-	-	-	-
42	62 yr M	Atherosclerosis. Contracted kidney	++	+++	++	+	++
43	63 yr F	Atherosclerosis. Contracted kidney. Bronchopneumonia	+++	++	+	+	-
44	64 yr M	Atherosclerosis. Softening of the brain	++	++	+++	-	+
45	65 yr F	Inanition. Dysentery	++	++	+	+	+
46	66 yr F	Uterine cancer. Dysentery	++	-	-	+	-
47	67 yr M	Inanition. Lobar pneumonia	+++	+++	++	+++	+++
48	67 yr M	Inanition. Dysentery	++	++	+++	+	-
49	67 yr F	Inanition. Dysentery	++	++	++	+	-
50	69 yr F	Pancreatic cancer. Dysentery	++	-	-	+++	+++